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SURGICAL PATHOLOGY

SYLLABUS,

BY

EDWARD H. NICHOLS, M.D.

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REPAIR OF TISSUES.

I. WOUNDS: A wound (trauma) is a break of the soft tissues of the body caused by violence. A wound may involve the skin (cutaneous) or only the tissues beneath the skin (subcutaneous).

Classification: Wounds are classified by their gross appearance, which varies according to the manner in which the injury is produced; hence incised, punctured, contused, and lacerated wounds. In incised and punctured wounds the edges of the wound generally can be approximated easily; in contused and lacerated wounds there may be extensive destruction of the surface tissues.

Complications: Various complications follow wounds of soft tissues. These complications may manifest themselves at once, or after a variable length of time, *i.e.*, may be immediate or secondary.

1. Immediate complications.

(*a.*) Hæmorrhage. The character of the bleeding depends upon the character, size, and location of the divided vessels, hence arterial, venous, capillary, or a combination.

(*b.*) Division of muscles or tendons, resulting in a mechanical loss of power in the injured muscles.

(*c.*) Division of nerves, resulting in a loss of power in the muscles supplied by the injured nerve.

(*d.*) Penetration of viscera, the results varying in accordance with the character of the organ injured. Emphysema may follow injury of the lung. If hollow abdominal vicera are wounded, extravasation of their contents and peritonitis may occur. Wounds of solid viscera may cause serious internal hæmorrhage.

2. Secondary complications are due chiefly to infection of wounded tissues by pathogenic microörganisms. If infection occur the microörganisms may multiply in the wounded tissues, produce a toxin, and cause various suppurative phenomena. They may cause a solution of subcutaneous tissue and an abscess, or they and their products may extend along the lymphatic vessels and produce lymphangitis and diffuse suppuration (phlegmon), or the infection may extend to adjacent blood vessels and cause coagulation of the blood (thrombosis), which may be followed by embolism and pyæmia; or a general infection, with toxæmia and septicæmia, may occur.

Aseptic and septic wounds: If a wound heals without infection by pathogenic microörganisms it is said to be aseptic; if microorganisms are present it is septic. Wounds seldom heal aseptically unless treated surgically.

Healing of wounds: In every wound the immediate result of the injury is a destruction of tissue. This destruction of tissue is followed immediately by the formation of exudation at the point of injury. In aseptic wounds the amount of exudate is proportional to the amount of injury, *i.e.*, to the extent of the wound. In septic wounds the amount of exudate is proportional to the amount of injury caused by the wound itself, and further to the amount of injury due to the action of the toxin produced by the microörganisms.

Following the exudate come certain proliferative changes, confined chiefly to the epithelium, connective tissue, and blood vessels about the injured area. Connective tissue and blood vessels form granulation tissue, which ultimately becomes dense scar tissue and replaces the tissue that has been destroyed.

The entire process of exudation and formation of scar tissue is spoken of as *repair of wounds*. The general steps of the process are the same, whether the edges of the wound can be approximated (incised or closed wound) or whether the edges cannot be approximated on account of extensive destruction of tissue (lacerated or contused or open wounds), but there are slight differences in detail, so that it is customary to describe the healing of aseptic closed and open wounds separately.

Repair of aseptic closed wounds, "First Intention": The process is seen in its simplest form and best studied in experimental lesions on animals, notably the ear or the tongue of a rabbit. The immediate result of the injury is hæmorrhage. In

a few hours the interval between the approximated edges is filled with an exudate of leucocytes, serum, and fibrin with some red blood corpuscles, the number of the latter depending upon the amount of hæmorrhage and diapedesis. The adjacent blood vessels are dilated. The exudate extends laterally for a considerable distance into adjacent tissues, and on the surface coagulates to form a crust, or "scab."

After a few hours, proliferation of epithelium and connective tissue adjacent to the wound begins. Adjacent epithelium proliferates, mitotic figures appear, and new epithelial cells begin to extend over the surface of the exudate beneath the scab. The connective tissue cells proliferate, young connective tissue cells, oval or polygonal, appear, and extend into the deep exudate. At the same time loops of young blood vessels are formed from adjacent blood vessels and project into the exudate.

This process continues for a variable time, depending upon the size of the wound, the character of the tissue, the amount of hæmorrhage, and the accuracy of approximation of the edges, until the space filled by inflammatory exudate is filled with newly formed loops of blood vessels and young connective tissue cells, "granulation tissue," while the surface of the wound is covered by proliferated epithelium. This process may be completed in seven days, but usually takes from three to five days more. As the proliferation advances, the exudate diminishes, and in experimental lesions may disappear about the seventh day.

Finally the epithelium is completely restored. The young connective tissue cells become spindle-shaped, with oval nuclei. Still later, intercellular fibrillar material appears between the cells, the blood vessels disappear, probably on account of pressure by the contractile connective tissue fibres, and the lost tissue is replaced by dense scar tissue, in which are few or no blood vessels, and the scar is covered by normal epithelium. The epithelium generally forms a thin layer, with parallel external and deep surfaces, and shows but little tendency to form papillæ. Sometimes, however, the epidermis sends long processes down into the scar, or islands of epithelial cells may become cut off in the depths of the wound. The entire process is completed in from ten days to two weeks, but the scar tissue continues to contract and become smaller several weeks, or even months.

In human wounds the process is the same, but owing to the extent of the wound and the complex anatomy the process

appears more complicated. The edges of the wound never are exactly approximated. The line of incision seldom appears sharply defined microscopically, probably because of injury due to manipulation during the operation. The inflammatory exudate extends to a considerable extent into adjacent tissues. When proliferation begins it extends to a considerable extent into the surrounding tissues, often for several centimetres on either side of the wound, and the deep line of incision cannot be accurately followed after the fourth or fifth day. The different layers of connective tissue, fascia, etc., take part in the proliferation to a varying degree. The process takes longer than in animals, and areas of lymphoid and plasma cells appear. In places small remnants of inflammatory exudate may remain after the greater portion of the wound shows advanced repair. If bits of fibrin remain, many giant cells appear. In human wounds the process of vascular scar formation takes about ten days, the time varying somewhat according to the size of the wound. The scar continues to contract and become less and less vascular for some months, or even for a year or more. Sometimes after several months microscopic section of a linear wound, which has healed by first intention, may fail absolutely to show any appreciable histological change, or at most may show a thinning of the epidermis along the line of incision, even although a white scar may be obvious to the eye.

The gross appearances correspond to the described histological process. At the end of a few hours the edges of the wound are sealed together by an adhesive layer of exudation, yellow or red, if there has been much hæmorrhage. In 24 to 48 hours this adhesive layer becomes hard and dry on the surface, and forms a crust or scab. The edges of the wound are somewhat swollen and reddened because of the presence of exudate and dilated blood vessels. The edges also are slightly hot and tender. If the edges are separated there is relatively little bleeding, since few new blood vessels are formed, and the tissues appear opaque and gelatinous from necrosis and infiltration with exudate. The space between the edges is filled with opaque exudate, partly coagulated. In from 3 to 6 days, if the crust is removed, the surface is seen to be covered with a thin layer of proliferated epithelium. The edges can be separated with difficulty and bleed freely if separated, the hæmorrhage coming from the granulation tissue. After about 10 days the line of the wound is

occupied by red scar tissue, composed of spindle-celled young connective tissue, in which are numerous young blood vessels. In the course of weeks or months the scar becomes white and narrow because of the disappearance of blood vessels and conversion of young connective tissue into dense connective tissue.

Repair of aseptic open wounds, "Second Intention": Healing by "granulation." As a result of injury there may be a more or less extensive destruction of the superficial and underlying tissues of the body, producing a wound so extensive that the skin edges cannot be brought together. In such cases the details of the reparative process differ from the process in closed wounds, although the general process is the same.

Almost always extensive hæmorrhage occurs, although if the wound is superficial this may be very slight. If it occurs the blood may coagulate upon the denuded surface. In a few hours an exudate is formed upon the surface of the wound, consisting of leucocytes, serum, and fibrin, and the exudate also extends to a considerable depth into the tissues about the wound. Adjacent blood vessels show moderate enlargement. In a few hours, by coagulation of blood and formation of fibrin, the wound is covered with a crust or scab.

Proliferation of epithelium and connective tissue and formation of new loops of blood vessels occur. The epithelium about the edge of the wound proliferates and grows into the exudate over the surface of the wound, and forms a film of proliferating epithelial cells. But epithelium has a limited power of proliferation, and, if the wound is extensive, may be unable to cover the surface of the wound for many weeks or months. If the wound remain open the epithelium may form a thickened, rounded layer of cells, depressed at the advancing edge.

Proliferation of connective tissue cells, with formation of oval or polygonal epithelioid or young connective tissue cells, takes place from the entire surface of the wound. These cells grow into and replace the exudate.

New blood vessels arise from blood vessels near the wound, and grow into the exudate. They are accompanied by the young connective tissue cells.

This combination of young blood vessels and young connective tissue cells forms "granulation tissue." Granulation tissue continues to form until the wound is filled to the level of the skin. Often the granulation tissue rises above the level of the skin if

the epithelial growth is insufficient to cover it ("exuberant granulation").

So long as the surface of the granulation tissue remains uncovered by epithelium a certain amount of exudate persists and is seen between the meshes of the granulation tissue. If the reparative process is prolonged, because of the size of the wound or from other causes, lymphoid and plasma cells are found in the tissue, often in large numbers. If the process is subacute, eosinophiles may be numerous.

If the wound is small and the reparative process takes but a few days the epithelium proliferates, grows in from the edge, and covers in the surface of the wounds. The young connective tissue cells become spindle-shaped, develop intercellular fibrillæ, and blood vessels become less numerous. At length (ten days in experimental lesions) the connective tissue becomes dense connective tissue, while blood vessels disappear. In that case the lost tissue is replaced by a mass of dense connective tissue covered with epithelium, *i.e.*, the wound is replaced by a contractile "scar." This process is seen best in experimental lesions.

On the other hand, if the wound is very extensive the epithelium may take months to cover over the surface of the wound. In that case the wound remains open, its superficial part is filled with granulation tissue, in which is exudate, lymphoid, and plasma cells, while the granulation tissue of the deeper part is converted into dense connective tissue, whose fibres run chiefly parallel to the base of the wound. Such a wound may be covered with a series of crusts, or, if the crusts are removed, with exudate.

The gross appearances correspond to these histological conditions. In an open wound, if very superficial, hæmorrhage may not be visible, *e.g.*, "barked" knuckles. The wounded surface is covered in a few hours with a clear or yellowish fluid, which coagulates in a short time. The fluid is exudate in which are more or less leucocytes, and this exudate coagulates and forms a thin film or crust. If the wound is deeper more or less hæmorrhage occurs. Then the wound in a few hours is filled with exudate and hæmorrhage. This mixture of blood and exudate coagulates and forms a reddish crust or scab. In two or three days is seen extending just beneath the edges of the crust a thin delicate white layer (proliferated epithelium). If the crust be removed a red, bleeding surface is exposed, which has a granular

appearance, hence the name "granulation tissue." The prominences of such tissue correspond to loops of blood vessels and their accompanying young connective tissue cells. The removal of such an adherent crust produces injury and necrosis of the granulating area and is followed by a slight inflammation, and in that way delays the process of healing.

If the wound heal promptly the epithelium extends over the surface of the wound and finally covers it with thin, pearly epidermis, the underlying granulation tissue becomes fibrillated and forms a somewhat raised, moderately firm red scar. After weeks or months this red color disappears as the connective tissue becomes denser, and the area of the wound is marked by a shining white, somewhat depressed mass of dense connective tissue, covered with epithelium, *i.e.*, a "scar." The scar is contractile, so that the area of the scar is always less than that of the original wound.

If the wound is extremely large, or the process of healing for any reason is delayed, the wound may remain open for weeks or months. In that case the edges of the wound are covered by a thin white film of proliferating epithelium, which still is unable to close the wound. The rest of the wound is filled with a reddish, granular, oozing layer of granulation tissue, which bleeds easily. This granulation tissue may project for a considerable distance above the level of the adjacent skin, "exuberant granulations." If this layer of granulation tissue is scraped off a layer of firm, pale, slightly vascular connective tissue (layer of dense cicatricial tissue) is seen. This layer is utilized in skin grafting.

Repair by "Third Intention": Occasionally in wounds in which there has been a considerable loss of tissue, it is possible after a few days to approximate the edges of the wound in such a way as to convert what was originally an open wound into a closed one. In such cases the early stages of repair are like those of the open wounds, *i.e.*, a proliferation of epithelium at the edges and a formation of granulation tissue at the bottom of the wound. If the edges then are closed by pressure or by approximation sutures, surfaces of granulation tissue, covered with a varying amount of exudate, are approximated in the deeper part of the wound, while at the surface edges of proliferating epithelium are brought together. In such cases under favorable circumstances the open wound is converted into a closed one, the granulation tissue from either side grows into and organizes the

exudate, the epithelium grows over and closes in the wound, and the later stages of repair are like those of a wound which is a closed one from the beginning.

Infected wounds: Closed or open wounds may become infected by various pathogenic microörganisms, either at the time the wound is made or at any later time before the process of healing is completed. If the wound is accidental it may be infected at the time it is produced; if the wound is an operative one infection may arise from surgically unclean instruments, sponges, dressings, or hands. If the wound is primarily aseptic it may be infected at a later stage by dressings which are not aseptic. Infection of surgical closed wounds is uncommon after the sixth day, because granulation tissue does not absorb as a nongranulating surface does.

So long as microörganisms continue to live and multiply in the tissues about a wound they produce a soluble toxin, which causes necrosis of tissue. Into the necrotic tissue comes exudation. Consequently effective repair cannot occur until the microörganisms are destroyed. The result of the infection of wounds is excessive destruction of tissue and a prolongation of the time required for healing.

Wounds may be infected by various microörganisms which produce suppuration, but the action of the different organisms results ordinarily in one of two sorts of changes in the tissues, *i.e.*, the organisms may produce (1) a localized necrosis, exudation, and solution of tissue about the point of infection, or (2) the infection may extend along the lymphatic spaces and vessels, and produce a more extensive and diffuse exudation with necrosis, but without marked solution of tissue. Infection by the staphylococcus aureus produces typical changes of the first kind; infection by the streptococcus produces typical lesions of the second.

Infected closed wounds: If a closed wound is infected the infection may be superficial or deep.

If the infection is by organisms of the type of the staphylococcus aureus, and is superficial, it commonly arises from infected sutures. In this case microörganisms are introduced along the track of the suture, multiply, produce a toxin, and cause necrosis of tissue and exudation along the suture, *i.e.*, a stitch abscess occurs (see Sutures). The infection frequently remains limited to the tissue about the suture, but may extend to and infect the entire wound.

If deep infection by organisms of the type of the staphylococcus occur the closed wound becomes practically an abscess cavity. Microörganisms multiply in the exudate and in the tissues about the wound, produce a soluble toxin, which causes necrosis and solution of tissue. Into the necrotic and dissolving area comes exudate, in which leucocytes predominate, *i.e.*, pus. If proliferation of connective tissue and vessels has begun, this granulation tissue may be infected and destroyed. About the area of solution new granulation tissue continues to form. This process of exudation, solution, and formation of granulation tissue continues until the contents of the infected wound are allowed to escape, or until the microörganisms die.

In closed wounds the products of such an infection usually are discharged early through the original wound, but if the edges of the wound have united early the amount of solution of tissue may be great. In this case, a condition is left exactly similar to the condition seen after an abscess has been opened, and the further steps of the healing are like those occurring in the healing of an abscess (see Abscess). The process may extend to the superficial layers of the wound and cause necrosis and solution of the approximated edges of the wound, and result in the formation of an open wound (ulcer). If this occur the further steps in the healing process are those already described under open wounds (*vide supra*), *i.e.*, granulation.

The gross appearances of closed wounds thus infected are characteristic. Pain, heat, redness, and swelling about the wound are marked. The edges of the wound are swollen, reddened, and gelatinous. In a short time gaps appear in the line of incision, through which is discharged yellow or bloody pus. The edges of the wound may become entirely necrotic, separate, and gape, disclosing a cavity lined with yellow or gray sloughs of necrotic granulation tissue. If these sloughs are forcibly removed they expose a raw, bleeding surface of granulation tissue. After the infection ceases an open wound is left similar in appearances to an uninfected open wound. Such a wound heals by granulation.

If a closed wound become infected by microörganisms of the type of the streptococcus the organisms multiply and produce lesions like those seen in lymphangitis (*vide* Diffuse Suppuration). In that case solution of tissue is less marked than in the type of infection just described, although some solution does occur.

Necrosis of tissue, however, is usually extensive, and leads to the formation of extensive sloughs, which must be disintegrated or removed before healing can be completed. The removal of this necrotic material usually results in the formation of a more or less extensive open wound, which heals by granulation, as already described.

The gross appearances of such an infection are characteristic. The wound is reddened, painful, swollen, hot, and tender. If the extension of the infection is along the lymphatic vessels, a distinct red line can be seen extending from the wound along the line of lymphatics. Tissues along the line of extension are swollen and tender. Groups of lymph nodes became swollen, tender, and may soften and form abscesses. If the condition is not relieved general infection, septicæmia, and toxæmia often occur.

If the extension is along connective tissue clefts ("cellulitis," "phlegmon") the tissues about the wound may show very extensive, diffuse redness, and heat and swelling, and be very painful. In this case extension of the infective process through the walls of adjacent vessels is common, and results in the formation of thrombi, with sometimes embolism and formation of metastases. Tissues about the wound usually become necrotic and form sloughs which must be removed before healing can take place. Removal of these sloughs leaves an open wound which heals by granulation.

Infected open wounds: Infection of open wounds may be due to organisms of the type of the staphylococcus or of the streptococcus.

Staphylococcus type: Infection by such organisms leads to a multiplication of organisms with a production of toxins, followed by necrosis, solution of tissue, and suppuration. The surface of the wounds breaks down, dissolves, and the area of the wound enlarges. After the infection ceases an open wound is left to heal by granulation.

Gross appearances: The edges of the wound are red, swollen, hot, and tender. The surface is covered with a profuse purulent discharge, beneath which is gray, necrotic granulation tissue. After the process ceases a wound, more extensive than the original one, is left to heal by granulation.

Streptococcus type: Infection by such organisms leads to multiplication of organisms with production of toxin and necrosis

with solution of tissue. As in closed wounds, the extension may extend along lymphatic vessels, or connective tissue clefts. In each case the conditions are like those already described under corresponding circumstances in closed wounds.

Gross appearances correspond to those already described under closed wounds.

Principles of treatment of wounds: From a consideration of the process of repair of wounds certain simple fundamental principles of treatment are obvious.

Surgical cleanliness is the most important factor, and is practically under control. This cleanliness applies to the field of operation, to the hands of the operator, instruments, sponges, sutures, dressing, and to all materials which in any way are brought into contact with the wound. If perfect surgical cleanliness (asepsis) is obtained, the amount of tissue to be repaired is dependent solely upon the size of the original wound. If pyogenic infection occurs, the destruction of tissue depends upon the extent of infection, and in all cases the extent of the wound and the length of time required to replace the defect are increased, to say nothing of the dangers of septicæmia, etc.

Avoidance of manipulation is also extremely desirable. In wounds in which long-continued or violent manipulation is carried on, the destruction of tissue extends very widely beyond the mere limits of a surgeon's incision, and in such cases the amount of tissue to be replaced is much greater than the mere incision would require. Even in incised wounds the extent of the reparative process beyond the line of incision is much greater than usually is appreciated. For this reason it is desirable for the surgeon to make free, sweeping incisions, rather than a series of little cuts.

Perfect and complete hæmostasis also is necessary to obtain rapid healing of wounds. The greater the amount of hæmorrhage between the edges of aseptic wounds the greater the length of time required for healing. The hæmorrhage separates the edges of the wound and increases the area to be organized by granulation tissue. Excessive amount of blood in a closed wound also furnishes an excellent culture medium for the growth of pyogenic organisms, if any are present.

Accurate closure and approximation of the edges of wounds in which an attempt is made to obtain primary union are essential. The approximation should affect not only the superficial edges,

but especially the deeper layers of the wound. Even with careful operators it is astonishing to see, on examining sections with a microscope, how very imperfect the closure of the wound is. The more accurately the epidermis is approximated the less the surface to be covered by proliferating epithelium. The quicker the wound is covered by epithelium the less the liability of infection. The more carefully the deeper layers are approximated and dead spaces are obliterated the less the amount of inflammatory exudate and blood to be removed and organized by granulation tissue, and the smaller the scar.

Aseptic protection of the wound is essential during the early days of the reparative process. The danger of secondary infection is over when the wound is covered by epithelium, and the exudate is entirely replaced by granulation tissue. The power of resistance to infection by a wound covered with granulation tissue is much greater than in the earlier stages before the formation of granulation tissue.

Fixation of wounded tissue also is essential to avoid continuation of injury to fresh or granulating edges of the wound. This fixation may be obtained in a variety of ways.

In regard to open wounds there are certain special precautions. Surgical asepsis is as desirable as in closed wounds, but perfect asepsis is not feasible in wounds which remain open for long intervals. The reason that extensive open wounds do not oftener become seriously infected is that healthy granulation tissue has a marked power of resistance to absorption of pyogenic organisms. On the surface of open wounds, in the early stages, masses of necrotic tissue ("sloughs") often are present. It is better not to attempt too vigorous removal of these, as their forcible removal leads to repeated traumatism of the young granulation tissue beneath them, with a consequent prolongation of the time of healing and increased danger of pyogenic infection.

In open wounds also it is desirable to keep the granulation tissue below the level of the advancing epithelial edge, as epithelium often is unable to cover over exuberant granulations.

In many cases of extensive open wounds the epithelium ceases to advance over the granulating area, and in such cases it becomes necessary to cover in the epithelial defect by small isolated grafts, plastic flaps, or Thiersch grafts.

Regulation of the blood supply always is desirable in open wounds. Venous stasis always appears to interfere both with the

formation of granulation tissue and with the advance of the epithelium. Prevention of venous stasis can be obtained by pressure, by removal of varicose veins, or by position.

II. Sutures: Various substances are used as sutures to approximate, support, and hold in position the edges of wounds. These artificial supports must be retained in position until the process of repair is so advanced that they no longer are needed. If the wound is superficial the ends of the suture remain visible and may be removed after repair is completed. Sometimes a superficial wound is closed by a suture which is buried in the edges of the skin, and such sutures usually are not removed. If the wound is a deep one deep sutures may be buried in the tissue and cannot be removed after healing is completed.

Sutures may be soluble in the tissues, or insoluble. Soluble sutures include catgut, plain or chromicized, and various animal tendons. Insoluble sutures include silk, silk-worm gut, silver wire, horse-hair, and celloidin.

The reaction produced in the tissues depends partly upon whether the suture is soluble or insoluble. The primary result of the introduction of sutures is the production of a minute wound. Soluble sutures at first act as a foreign body, but after a time are dissolved, absorbed, and leave a gap in the tissue, which is filled by scar tissue. Insoluble sutures act as a foreign body indefinitely, unless removed, and finally are surrounded and encapsulated by scar tissue.

Catgut may be taken as the type of soluble suture; silk as the type of insoluble.

Catgut sutures: Introduction of the suture causes a minute wound. Along the track of the suture and extending some little distance into the surrounding tissues comes an exudation. The gut soon becomes swollen and fibrillated. Leucocytes appear between the fibrillæ and at the periphery of the suture. Wherever they appear there is marked solution of the suture, so that it becomes much reduced in size. About the inflammatory area a layer of granulation tissue is formed, well marked on the third day. In this granulation tissue giant cells are few or wanting. In about ten to fourteen days the gut is entirely absorbed, the exudation disappears, and the space occupied by the suture is filled by granulation tissue. At length this granulation tissue becomes dense scar tissue.

Gross appearances: At first the tissue about the suture shows slight swelling and redness. After a few days, if the suture is removed, it leaves a small circular hole from which serum and a few leucocytes can be expressed. At the end of about ten days the external portion easily separates at the level of the skin, due to the fact that the buried portion of the suture is dissolved, and a tiny red circular scar of granulation tissue shows the former site of the suture. In time this becomes a minute white scar, or sometimes may disappear.

Silk sutures: As in the case of catgut the introduction of the suture causes a minute wound, which in a few hours is filled with exudate. The exudate extends for some distance into surrounding tissues. The leucocytes appear in large numbers between the fibres of the silk. In about forty-eight hours proliferation and formation of granulation tissue about the suture is marked. This granulation tissue replaces the exudate and may even extend between the fibres of the silk. In this young connective tissue appear many giant cells, which generally lie in close approximation to individual fibres. Ultimately the exudate disappears, leaving the suture encapsulated and enmeshed in dense fibrous tissue. Giant cells may persist for months or years.

Gross appearances: Superficial silk sutures generally are removed at about the tenth day. When removed they leave a small circular wound from which serum and a few leucocytes can be expressed. This wound is closed by granulation tissue in a few hours. At length, only a minute white scar is left.

Sutures of chromicized catgut, or kangaroo, or other tendons, are much less soluble than catgut, but generally are ultimately absorbed. At times, however, they may persist for years. In such a case it is probable they are encapsulated. Silk-worm gut, silver wire, and horse-hair are insoluble and not fibrillated, so that no infiltration or enmeshing of the suture can occur.

Either silk or catgut sutures may be septic. In that case the track of the suture may become infected with microörganisms, and become a minute abscess cavity. The infection may remain confined to the track of the suture, or may extend and infect the entire wound (see Infected Incised Wounds).

No ideal or perfect suture has as yet been discovered. A suture which is perfectly satisfactory for one purpose may be very unsatisfactory for another.

III. Wounds of the intestines: When any portion of the intestinal tract is wounded, it is essential that the wound be closed at once in such a way as to render the wall of the intestine water-tight as soon as possible, so as to avoid leakage of the infectious contents. Consequently many methods have been devised to give as perfect as possible mechanical closure, which, however, is never absolutely perfect; and the serous surfaces of the cut edges always are approximated, because, if mucous membrane surfaces are brought together, repair does not begin until the epithelium has been sloughed off; while, when the external (serous) surfaces are approximated, the production of fibrinous exudate is very rapid, and in a very few hours makes the wound water-tight, provided too much mechanical tension is not put on the wound. Many methods of suture have been devised to close the intestine. In some cases the suture may penetrate all the coats of the gut, but these sutures as a rule are applied merely to give fixation of the wounded edges. The sutures which approximate the serous surfaces of the intestine should not extend from the lumen of the intestine to the peritoneal cavity, for if they do they make a wound connected with the infected intestinal canal, and infection along the suture may lead to infection of the general peritoneal cavity. The best suture is one which gives the strongest and most perfect immediate mechanical closure of the wound without allowing any connection of infected intestine with peritoneal cavity, and also gives the most perfect approximation of the external serous coat without diminution of the calibre of the intestine. It may be said that without doubt the best suture material on the whole is silk or celloidin, as animal sutures soften so early that they do not maintain perfect approximation until the wound is completely organized. Mechanical devices should be used only for special clinical reasons. The process of repair is the same no matter what mechanical method is used, but the process described is such as is seen after suture.

The reaction produced by the suture corresponds to that already described, and will be ignored in describing the process of repair. Serous surfaces are approximated because of the large amount of exudate formed from serous surfaces, which quickly seals the wound hermetically.

The wounded area is filled quickly with exudate and all layers of the gut are infiltrated. The invaginated edges necrose and the endothelium between the approximated edges, and for a con-

siderable distance on either side of the wound, disappears. Exudate extends over the serous surface for a considerable distance beyond the limits of the wound (aseptic local peritonitis). Formation of fibrin quickly seals the inverted serous surfaces and prevents leakage of intestinal contents. In a very few hours (24) there appears extensive proliferation of connective tissue, especially of the subserous connective tissue, and a formation of new vessels. This granulation tissue extends into the exudate from either side, the exudate disappears, and by fusion of the layers of granulation tissue the external surfaces of the gut are joined by granulation tissue. This process may be completed as early as the seventh day, although union generally is not firm before the tenth day. Meanwhile the inverted edges become necrotic and dissolve, leaving an ulcer on the mucous surface of the gut. The outer surface of the gut, for a distance of several inches, may be covered by exudate, which finally is converted into granulation tissue. At length the granulation tissue between the inverted edges may become dense fibrous tissue (intestinal scar). The internal ulcer heals, and there may be a partial reproduction of intestinal glands. The external exudate becomes organized. This organization of the external exudate may cause adhesions to other serous surfaces. As the granulation tissue is converted into dense fibrous tissue, some contraction may occur, and, unless provided for, may lead to reduction of the calibre of the lumen of the gut.

Gross appearances: For the first day or so the inverted edges form a marked projection into the lumen of the gut. Between the serous surfaces is a layer of adhesive exudate. The outer surface of the gut is injected and covered with a layer of fibrinous exudate. In a few days more the invaginated edges begin to melt down and disappear, and a more or less raised ulcer appears on the inner surface of the gut.

The wound is closed by a red line of organizing exudate, while the external exudate becomes firmer, and, possibly, firmly adherent to adjacent viscera from the presence of granulation tissue. In the course of a few weeks the internal surface is covered by more or less complete mucous membrane, the wound is closed by a barely visible white scar, while the external surface of the gut is opaque and dull from the presence of fibrous tissue. Ultimately it may be impossible to find the point of incision.

The process of repair in wounds of the intestine usually is

more rapid than in wounds of the surface of the body. The wound is sealed by fibrinous exudate within a very few hours, although, of course, the fibrin can easily be displaced under great tension. In many cases the organization of the exudate is completed in seven days, although it is to be remembered that granulation tissue at this time still is very fragile. Rapidity of union is favored by accurate approximation of serous surfaces and by avoiding manipulation as far as possible, so as to avoid traumatism of the approximated edges.

IV. Repair of tendons: Tendons, aponeuroses, and ligaments are special types of connective tissue. What has already been said about the repair of connective tissues in wounds applies in a general way to the repair of tendons, but tendons are connective tissue with a special function and a special structure, and the details of the process of repair vary somewhat from the process as seen in, *e.g.*, subcutaneous connective tissue, and deserve especial mention. To understand the process of repair it is necessary to bear in mind the anatomy of a normal tendon.

Tendons are composed of the densest sort of fibrous tissue arrayed in parallel bundles, closely connected, with relatively few elastic tissue fibres. Surrounding the tendons is a layer of loose areolar tissue (the peritendineum) from which septa run into the tendon, dividing it into large (secondary) and smaller (primary) bundles of dense fibres. These dense fibres appear, under the microscope, wavy from contraction, and anastomose more or less with each other. Between the fibres are cells which on long section are oval or rectangular, but on cross section are stellate, and are united to other similar cells by processes, thus separating the fibres into bundles.

When a tendon is divided there always is considerable retraction of the divided ends. This is due partly to contraction of the muscle of the tendon and partly to contraction of the fibres of the tendon itself. The peritendineum seldom retracts to the same extent as the tendon but becomes markedly fibrillar and folds over the retracted end of the tendon. Into the interval between the retracted tendon ends comes an inflammatory exudate, with perhaps some hæmorrhage. The mesh of the peritendineum is filled with exudate, but the exudate extends only a little way into the cut ends of the tendon itself. Very early there begins a very rapid proliferation of connective tissue cells from

the connective tissue of the mesh of the peritendineum, not only between the retracted ends of the tendon, but also from the peritendineum outside the tendon ends, thus forming a spindle-shaped swelling much like the callus of a fracture. At the same time new blood vessels are formed. In this spindle of granulation tissue intercellular fibrils appear very early to an extent much more marked than in ordinary connective tissue. The cells of the tendon take very little part in this process of proliferation, and the original dense fibres of the tendon not at all. There is, however, marked proliferation of the connective tissue cells of the connective tissue septa of the tendon, which extend between the dense fibres of tendon proper. As the proliferation continues the exudate disappears, and finally the cut ends are joined by a spindle of granulation tissue. The blood vessels disappear very early, granulation tissue fibrils are formed in large amounts, and a spindle of dense, fibrous tissue joins the cut ends. In time the new intercellular fibrils cannot be distinguished from the original tendon fibres, the new tissue becomes of the same size as the original tendon, and cannot be distinguished by the naked eye from the uninjured tendon.

The time required for the process is variable, depending upon the size of tendon and upon the amount of separation. The formation of completely organized, dense, fibrous tissue in smaller tendons is completed in about two weeks. In larger tendons the process is somewhat longer. The tendon practically always is sufficiently regenerated to allow passive motion in about three weeks.

In some cases tendons fail to unite or may unite imperfectly. If the cut tendon ends are too widely separated, the connective tissue of adjacent connective tissue may interpose, and the gap be filled with ordinary scar tissue instead of with connective tissue arising from the peritendineum; or again, even if the two tendinous ends become united with new tendon derived from the peritendineum, the new tissue may become adherent to the new connective tissue of the adjacent skin, etc., so that imperfect function results. Also, where several tendons are divided in one wound, *e.g.*, in accidental wounds about the wrist, especially if the tendons are divided in some place where the tendon sheaths are not sharply defined, the newly-formed tissue between the ends of adjacent tendons may unite into one common mass, thus leading to very imperfect function. In the same way, even when only

one tendon is divided, it may adhere to the connective tissue wall of its sheath. In the case of tendons lying in sharply defined sheaths, a large amount of separation is possible. In cases where several tendons lie close together, less separation is possible. In such cases it often is better to fill the gap by some one of the many methods of splicing the tendon, in order to be sure that the line of the tendon is maintained by peritendineum from which the new tendon is to be formed.

V. Fractures: If a bone is fractured, usually the ends of the bone are more or less displaced, fragments of bone may lie loose in the tissues, there usually is more or less stripping of the periosteum, with crushing of adjacent soft parts, and some hæmorrhage. After a few days a fusiform mass (the "callus") is formed about the fractured ends, and persists for a time. After the ends of the bone are firmly united the callus disappears more or less completely, and, if the ends of the bone are accurately approximated, the external appearance of the bone becomes normal.

The process of repair of fractured bone is best studied in bones in which a loss of tissue has been produced without dislocation of fragments, *e.g.*, by drilling a small hole vertically into the shaft of a bone. In this way the reparative process is uncomplicated. Later, the more complex process seen in ordinary fractures easily is understood. The process as seen in experimental drill-holes is described first, then the more complicated process of complete fracture.

Experimental fracture (drill-hole): As a result of the injury, hæmorrhage occurs, followed in a few hours by more or less exudate of leucocytes, serum, and fibrin.

By the second day proliferation of connective tissue and of the endothelium of blood vessels begins. This proliferation of connective tissue occurs external and internal to the cortex of the bone. The external proliferation arises from the periosteum, and results in the formation of a spindle-shaped mass of connective tissue, in which are young blood vessels derived from neighboring blood vessels, about the injured point (early "external callus"). The internal proliferation arises from the connective tissue of the marrow, which lies next to the cortex of the bone (endosteum), and so appears as a mass of young connective tissue about the edges of the drill-hole ("myelogenous callus") and, if the

injury has been sufficiently deep, from the endosteum of the bone opposite the drill-hole. At this period remnants of exudate persist. Fragments of necrotic cortical bone carried in by the drill may be seen

By the fourth day a homogeneous intercellular substance ("osteoid tissue") appears between many of the granulation tissue cells of the internal callus. In this osteoid tissue, at a later period, lime salts are deposited; *i.e.*, it represents the earliest outline of new bone trabeculæ. In this homogeneous material are included some of the connective tissue cells which later are to become "bone corpuscles." Certain connective tissue cells at the periphery of these new trabeculæ deposit successive layers of osteoid tissue, *i.e.*, act as "osteoblasts."

Formation of osteoid tissue also takes place in the external layer of granulation tissue of the external callus.

By the end of a week the exudate has nearly disappeared. Trabeculæ are well developed both in external and internal connective tissue. The spaces between the trabeculæ are filled with young connective tissue cells and young blood vessels. Many osteoblasts lie about the trabeculæ and osteoclasts appear. The external callus covers in the wound produced by the drill. The internal callus may partly or completely fill the medullary canal at the point of injury. The cortical ends of the bone take no part in the proliferative process, but the hole in the cortex becomes filled with granulation tissues which extend from the external callus inwards, and from the internal callus outwards.

At the end of about four weeks both external and internal callus are composed entirely of newly-formed trabeculæ, and the mass of fibrous tissue which filled the drill-hole is converted into young bone and unites the separated cortical ends. The young trabeculæ, as a rule, lie at right angles to the original shaft. Many osteoblasts are seen lying close to the trabeculæ.

Ultimately the external callus is absorbed and largely disappears. The internal callus also may be absorbed, and the marrow canal restored. The trabeculæ between the separated cortical ends remain, become closely attached to the separated cortical ends and restore the line of the cortex, and repair is completed. In time the trabeculæ rearrange themselves, so as to lie parallel with the line of cortical bone.

Observe that in experimental drill-holes the transition from

granulation tissue to bone is direct, without the formation of cartilage as an intermediate step.

Complete fractures: The general process is the same, but there are slight modifications in detail. In complete fractures the ends of the bones are more or less dislocated, since exact approximation of the ends of the bones seldom occurs. Injury to soft tissues also is more extensive.

When proliferation begins, it arises internally from the connective tissue of the marrow ("medullary callus"), externally both from the periosteum and from the connective tissue of adjacent soft parts ("external callus"). Consequently the external callus appears relatively much larger than in drill-hole fractures, and forms a large fusiform mass which includes the dislocated ends of both fragments. This external callus at first is composed entirely of granulation tissue, and no distinction can be made between the portion which has arisen from the periosteum and that which has arisen from the soft tissues.

When trabeculæ appear they may form directly from granulation tissue, as occurs in drill-hole fractures, but commonly in complete fractures the granulation tissue nearest the shaft of the bone becomes dense fibrous tissue, and then is transformed (metaplasia) into cartilage. As this metaplasia of connective tissue extends farther into the fibrous tissue the deeper layers of cartilage are converted into bone, and trabeculæ and marrow spaces appear. The process of repair in the internal callus resembles that described under drill-hole fracture.

Ultimately, there comes more or less absorption of the trabeculæ of the callus, and a restoration of the shaft to approximately its original condition. If the dislocation of the ends is excessive, much of the callus always persists; *i.e.*, excessive callus indicates malposition of fractured ends. The time required to repair any given bone depends upon the size of the bone, the accuracy with which the ends of the bone are approximated, and the care with which they are immobilized.

In some cases bony union may be delayed for many months or may never occur. The cause of this condition is not clear. Sometimes it appears to be due to mechanical causes (by inclusion of soft tissue between fractured ends), at other times it is attributed to "constitutional" causes. In some cases the formation of osteoid tissue seems complete, but for some unknown reason no deposition of lime salts occurs.

In some cases the ends of the bone are united by dense fibrous tissue only, so that a flail-like condition persists ("syndesmosis"). Or one fractured end may enlarge and form a false socket, while the other end forms a false head and a false joint ("pseudo-arthrosis") is formed. In such cases the two ends usually are surrounded by a capsule of dense cicatricial tissue. The articulating ends of the bones generally are covered with dense fibrous tissue, not with cartilage. If two adjacent bones are broken, *e.g.*, both bones of the forearm, there may be fusion of the calluses, and the two bones finally be firmly united ("synostosis").

Gross appearances: After a fracture the soft tissues about the fracture soon become swollen and tense from the pressure of exudate. In a few hours blebs and bullæ, containing a clear serous or hæmorrhagic fluid, may appear, due to elevation of the superficial layers of the skin by the fluid exudate. If the injury to soft tissues is severe they may be colored bluish at once by extensive subcutaneous hæmorrhage, or the tissues may appear black and blue only after several days, due to deep hæmorrhage and disintegration and diffusion of blood pigment.

After the exudate diminishes the tissues about the site of the fracture are thickened and form a spindle-shaped mass (granulation-tissue external callus), at first firm and elastic, but not bony. After about two weeks the callus obviously becomes harder and more sharply defined (ossification of callus), and the fragments which at first were freely movable are much less so, or move only under strong pressure. After a variable number of weeks, depending upon the site and severity of the fracture, mobility entirely disappears, although the thickening due to the callus persists. At this time the bone is strong enough to bear weight. In the course of months the callus progressively becomes less (absorption of external callus), and finally becomes very small. Complete absorption seldom occurs, but the amount of callus which persists is proportional to the amount of deformity. Persistence of a large callus almost always indicates malposition of the broken ends.

Complications: Certain complications of fractures occur. Blood vessels, nerves, or tendons may be torn by the fractured ends of the bone at the time the injury occurs. If large blood vessels are injured severe hæmorrhage into tissues, or even gangrene of an extremity, may result. If nerves are injured paralysis or trophic changes may occur. Tendons may be

severely injured, and become adherent to tendon sheaths as repair takes place, so that serious disability is produced.

If the cutaneous tissues are wounded at the time the break occurs, an open wound may be produced leading to the point of fracture (compound fracture). If infection of the wound by pyogenic organisms take place, serious or fatal suppuration of the bone may take place (traumatic osteomyelitis, *q.v.*).

Principles of treatment of fractures: Depend upon the above-described conditions.

The first essential is as perfect as possible approximation of the fractured ends. The more perfect the position of the fracture the smaller the external callus and the shorter the time required for completion of repair of the bone. The more perfect the position, the less is the interference with the soft tissues, and in all fractures it is to be remembered that there is injury not only of bone, but of soft tissues. As regards the reduction of the deformity it should be borne in mind that attempts at reduction more than two weeks after injury are likely to give poor results. During the earlier stages the callus is soft and not ossified. After the second week bone formation is well advanced, the ends of the bone are included in the spindle-shaped callus, are not freely movable as at first, and forcible correction causes injury to the newly-formed bone and prolongation of the process of repair.

Perfect immobilization also is essential. The less the callus is interfered with, the greater is the rapidity of repair. In case of fracture about tendons or into the articular surfaces of joints, other mechanical problems enter in, so that early mobility may be necessary and the importance of rapid ossification may be secondary.

VI. Repair of muscle: After a wound of striated muscle there comes, as in all injuries of the soft tissue, an inflammatory exudate. In the course of a few hours there arises a new growth of granulation tissue from the adjacent connective tissue and also a peculiar series of changes in the muscle itself. Some of the muscle fibres next to the wound become necrotic; they are invaded by polynuclear leucocytes and endothelial cells, dissolved, and removed. In some of the other muscle fibres there occurs an increase in the number of the nuclei, which arise not by mitosis, but by direct nuclear division. These nuclei arrange

themselves in the ends of the muscle fibres, and, instead of having a mural arrangement like that of the nuclei in normal muscle fibres, are situated in the middle of the fibre. The fibre itself loses its striæ, and becomes more or less fibrillated longitudinally. The greater portion of these cells finally disappear, so that in the granulation tissue scar only an occasional club-ended fibre is left, and the defect in the muscle is replaced by granulation tissue, which ultimately becomes scar tissue. If the ends of the muscle are accurately approximated the scar is a small one and interference with muscle function is slight. If the ends of the muscle are widely separated, there may be great impairment of function.

VII. Repair of the heart: In wounds of the heart the muscle fibres take no part in the process of repair, but the defect is filled by granulation tissue, which finally forms a scar. Adhesion to the pericardial walls is common.

VIII. Repair of blood vessels: Wounds of vessels of large size present a condition somewhat different from that of wounds of other tissues, since the walls contain no small vessels except in the adventitia coat, so that the early adhesion of the edges of the wound is produced not by an inflammatory exudate in the ordinary sense of the word, but is due to fibrin which arises from the circulating blood in the vessel itself. The conditions vary somewhat in arteries and veins, and with the character of the injury, *i.e.*, whether there is complete division of the vessel, or a lateral wound, or a rupture of the internal coat.

In arteries complete division of the wall by a sharp instrument of course leads to violent hæmorrhage, which may cause death in a short time. In complete division of an artery by tearing or by similar violence, however, extensive hæmorrhage as a rule does not take place, and may be absent even in clean cuts, because the ends of the vessel retract into the surrounding tissues, while the walls of the vessel become occluded by the formation of a clot, composed of fibrin derived from the blood in the vessel, enclosing red blood globules and a few leucocytes. In wounds in the wall of an artery the hæmorrhage takes place into the soft tissues about the point of injury, and coagulates, so that finally the edges of the vessel wound are sealed together by a layer of fibrin.

Within the lumen of the vessel may be simply a thin peripheral clot at the point of injury, covering the wound; or in other cases, especially if the endothelium is extensively injured, a thrombus may form, of such size as to occlude the vessel. In ligature of a completely divided vessel a clot forms at the point of ligature, the size of which is variable, depending upon the rapidity of circulation, the amount of injury to the endothelium, and the perfection of asepsis.

After the formation of the thrombus the later stages are like those of any wound; *i.e.*, the clot, which is essentially an inflammatory exudate, in which red blood globules are overwhelmingly predominant, becomes converted into organized tissue. If the clot is a small one, situated peripherally, the surface may be covered by newly-formed endothelial cells, while the deeper layers of the clot are replaced by newly-formed connective tissue derived from the media and adventitia. If the clot is of large size and completely fills the lumen of the vessel, the surface of the clot toward the blood stream is covered with endothelium growing from the walls of the vessel, while the clot itself becomes organized by granulation tissue. The lumen of the vessel beyond the point of obstruction slowly is diminished in size by an obliterative endarteritis.

In some cases, where the wound in the vessel is a lateral one, a large clot forms about the point of injury, pushing apart surrounding soft tissues, until the pressure becomes so great that no further hæmorrhage takes place. The effused blood coagulates, and finally the periphery may become organized by granulation tissue arising from adjacent connective tissue. In some cases the centre of this area may remain patent and contain fluid blood, connected with the circulating blood in the patent vessel through the interval in the wall made by the wound, thus forming a false traumatic aneurism.

In complete division of veins the divided ends usually are filled with a blood clot which becomes covered by endothelium, while the clot itself comes to be replaced by dense scar tissue derived from the media and adventitia. In some cases the amount of terminal clot, however, is exceedingly small. In case of a lateral wound of a large vein, it often is possible to prevent severe hæmorrhage by ligaturing the wound in the vessel. In that case the inner wall of the veins is puckered by ligature, and may be covered with a thin, peripherally placed clot, which may become

organized without the formation of an obstructing and obliterating thrombus.

In all lateral wounds of vessels absolutely perfect asepsis is essential if one expects to obtain healing without complete thrombosis and obstruction. The presence of even a slight amount of infection is practically certain to cause sufficient injury to the endothelium to produce complete obstruction. In suturing of vessels it is said that the projection of perfectly aseptic sutures into the lumen of the vessel through the endothelium does not necessarily produce thrombosis, but that in many cases the sutures are very early covered with new endothelium.

IX. Peripheral nerves: Section or destructive injury of a peripheral nerve causes an immediate traumatic local degeneration of the nerve at the point of injury. This is followed by a degeneration throughout the extent of the nerve peripheral to the point of injury, and a degeneration of the fibres proximal to the point of injury, extending no farther than the first few nodes of Ranvier. There also occur changes in the cells of origin of the degenerated nerves, resulting in an effacement of the granular structure of the nerve cell body, with displacement of the cell nucleus to the periphery of the cell, the so-called "axonal reaction" of Nissl.

Following the degeneration occur regenerative changes in the nerve, which may lead to a restoration of function. The extent to which this regeneration may occur depends somewhat upon the amount of injury to surrounding soft parts. If the injury is one which destroys the integrity of the nerve fibre, without destroying the continuity of the nerve sheath, *e.g.*, crushing injuries, the regeneration of the nerve is more rapid and certain. If the nerve is cut across and the ends are sutured together, regeneration is more likely to occur than it is if the ends retract and become widely separated, or if suppuration occurs so that the ends are separated by a wide zone of granulation tissue. If the peripheral end of a nerve is entirely removed, as, *e.g.*, in an amputation, a peculiar partial regeneration of the proximal portion may occur, resulting in an "amputation neuroma."

After the receipt of an injury there comes a traumatic degeneration of the nerve in the immediate vicinity of the injury. The amount of this degeneration depends upon the character of the

injury, being, *e.g.*, slight in a clean-cut wound and more extensive after a crush. Immediately after this change there comes a secondary ("paralytic") degeneration of the nerve, extending in either direction from the point of injury. On the central side of the injury the degeneration extends upward to the nearest nodes of Ranvier. On the peripheral side the degeneration extends throughout the entire extent of the nerve.

The degenerative changes produce a fragmentation and fibrillation of the axis cylinder, and a fragmentation of the medullary sheath. Very early there also arises marked proliferation of the cells in the sheath of Schwann.

The regenerative process begins after the degenerative process. It is difficult to say just how the new axis cylinders are produced, there being dispute upon this point; but the new axis cylinders extend gradually into the peripheral end. Most observers believe that the process of growth is like that in embryological development, *i.e.*, a constant peripheral growth. Others believe that the new formation is, partly at least, the result of activity of cells in the sheath of Schwann. As a practical matter, the new fibres in the adult always arise from the central stump and extend peripherally along the track of the original nerve.

The new fibres, as they arise from the central stump, tend to split into bundles of small neuro-fibrils, of which the original nerve is supposed to be composed. The direction of the new fibres may be modified by various mechanical obstructions, and also by an apparent attraction of the distal nerve remnant for the proximal nerve fibres. The fibres at first grow in the interstices between the cells of the scar ("neurotization of the scar"), which lies between ends of the nerve. If the scar between the ends is too dense the new fibres may grow into the tissues in various directions, and never may be able to get into contact with the peripheral stump. In such cases no restoration of nerve function takes place. The tendency of the proximal axones to join the peripheral stump can be favored by various mechanical means, *e.g.*, catgut sutures along the tract of the nerve, or hollow tubes, or neuro-plastic flaps. Regeneration is obstructed by secondary infection with excessive formation of granulation tissue.

The rate of generation varies somewhat, but is approximately at the rate of 1 mm. per day.

Central nervous system: As regards regeneration or repair of injuries to the central nervous system, while theoretically possible to a very slight degree, the amount of regeneration is so slight as to be of no surgical importance. The cause of the lack of power of central nerves to regenerate is obscure, but it is claimed to be due to the fact that the central nerve fibres do not possess a sheath of Schwann, which is essential in some way to the new formation of axis cylinders, and also to the fact that the neuroglia fibrils offer a mechanical obstruction to the advance of nerve fibres.

SUPPURATIVE EXUDATIONS.

Under certain circumstances leucocytes may be present in enormous numbers in exudate. The exudate then appears thick, yellow, and creamy, and is called "pus." An exudate of this sort can be produced experimentally by injecting certain chemical substances (*e.g.*, croton oil) into the tissues of animals, but in human beings it practically always is due to infection by certain microörganisms ("pyogenic bacteria"). These bacteria produce certain soluble substances which attract leucocytes in enormous numbers, and a cavity may be formed in which leucocytes and tissue products are contained. Such a cavity is an "abscess," and may open upon the surface of the body by a narrow channel ("sinus"). If the surface of such an abscess cavity is destroyed so that an open sore is produced, an "ulcer" is formed. The process just described is spoken of as "frank" suppuration.

In other cases the organisms produce extensive necrosis and suppuration, without marked solution of tissue. The organisms then tend to extend along the lymphatic vessels ("lymphangitis"), or along the connective tissue septa ("phlegmon," "cellulitis"). In that case the process is spoken of as "diffuse" suppuration.

Typical frank suppuration is produced by the staphylococcus aureus. Typical diffuse suppuration is produced by the streptococcus.

Acute suppurative inflammation may extend to and involve the adjacent blood vessels, causing coagulation of the blood ("thrombus"). Such a thrombus contains pyogenic organisms. Fragments of such a thrombus may be torn off (embolus) and carried into the general circulation, and lodge in distant parts of the

body. Such emboli, since they contain infected organisms, may act as the secondary foci of suppuration ("metastases").

Abscess formation: Frank suppuration of the type produced by staphylococcus aureus. The furuncle and carbuncle may be taken as examples of this type of inflammation.

Furuncle: A furuncle is an acute suppuration of the skin and subcutaneous tissues, which develops about a hair follicle or sebaceous gland.

Under certain conditions the staphylococcus aureus may obtain access to the subcutaneous tissue through the unbroken skin along a hair follicle. Having invaded the tissues the coccus produces a soluble toxin which causes necrosis of adjacent tissues. Into the necrotic area comes exudation. By action of the toxin the necrotic tissue is dissolved, so that a cavity is formed containing exudate, bits of necrotic tissue, and very little fibrin, *i.e.*, "abscess." About this abscess cavity comes proliferation of adjacent connective tissue and formation of new vessels. This layer of granulation tissue is the so-called "pyogenic membrane." If the cocci continue to develop this pyogenic membrane may, in turn, become necrotic and dissolve, so that the abscess enlarges, while granulation tissue continues to form about the enlarged abscess. If the process last for any length of time lymphoid and plasma cells are seen in the surrounding granulation tissue. The process continues until the cocci lose their virulence, or the contents of the abscess are evacuated. After the contents are removed granulation tissue grows into and fills the cavity, while the epithelium about the opening proliferates and covers it. At length the granulation tissue is converted into dense fibrous tissues, and forms a scar.

Gross appearances: A pustule appears about the base of a hair. In a few hours the pustule is surrounded by a circumscribed dark red swelling, hot and painful. From the fifth to the seventh day a yellow softened area appears at the apex of this swelling, *i.e.*, the furuncle "points." Through this opening is discharged a mass of yellow, necrotic material ("slough"), leaving a cavity lined with granulation tissue. In a few days after the furuncle is evacuated the cavity is filled by granulation tissue, while the surface is covered with proliferated epidermis. The entire process takes about two weeks. The granulation tissue finally is converted into dense fibrous tissue, and forms at first a reddened and later a white scar.

Carbuncle: A carbuncle is a suppuration of the skin and subcutaneous tissues, similar in character to the furuncle, but usually more extensive and severe, and modified somewhat by the anatomy of the skin in certain parts of the body. In some portions of the body the subcutaneous tissues are very thick, and the hair follicles extend only part way through it. Extending downward from the base of these hair follicles are clefts in the true skin. These clefts are filled with fat ("columnæ adiposæ"). From the sides of the base of these columnæ strong bands of fibrous tissue extend to the underlying fascia, binding the skin firmly to the fascia. The staphylococcus aureus is the common cause of carbuncle.

The staphylococcus probably obtains access to the subcutaneous tissues along the hair follicles. Having reached the subcutaneous tissue, the process is the same as in the furuncle — necrosis, infiltration with leucocytes, and solution. Because of the firm adhesion of the skin to the underlying fascia by the fibrous bands already described, the extension of the process laterally is not easy, so it extends along the line of least resistance, *i.e.*, into the columnæ adiposæ to the base of the adjacent hair follicles, and from these to the surface of the skin. In this way numerous secondary areas of suppuration are formed upon the surface. By continuation of this process and fusion of adjacent foci a large central area of necrosis is formed, surrounded by secondary smaller areas, due to extension from below.

About the necrotic area comes marked proliferation with production of granulation tissue. After the process ceases to extend, the necrotic tissue dissolves and is separated from the underlying granulation tissue, leaving a more or less extensive, frequently very deep ulcer. Ultimately this ulcer is filled in by granulation tissue, while the surface of the ulcer is covered by proliferated epidermis.

Gross appearances: The carbuncle appears as a diffuse, brawny swelling, generally upon the neck or back; bluish-red at the centre, and a brilliant red at the periphery. In the centre appear numerous yellow, soft points usually one large one in the centre, surrounded by small ones. Ultimately these yellow areas enlarge and coalesce, forming a large yellow slough in the centre of the area. After some time this slough dissolves and is cast off, leaving a large granulating wound which closes by granulation.

Diffuse suppuration: "*Phlegmon*," "*Lymphangitis*," is a diffuse acute suppuration of the skin and subcutaneous tissues, generally secondary to some wound of the skin. It generally is due to infection by the streptococcus pyogenes.

The infection extends along the line of lymph vessels and connective tissue clefts. The organisms produce necrosis and exudation. The subcutaneous tissue early becomes necrotic, often over enormous areas, but solution of the tissue is much less marked than in abscess formation, although some localized areas of solution may appear. If the infection is mild the exudation soon is absorbed, the necrotic tissue is dissolved and absorbed, and the area occupied by the necrotic tissue becomes filled with granulation tissue, which ultimately becomes dense fibrous tissue, so that the skin becomes firm, hard, and adherent, and may cause much impairment of function of underlying muscles. Sometimes the necrosis involves the superficial layers of the skin, and the necrotic area may be separated from the underlying granulation tissue in the form of an extensive slough, leaving behind a wound which closes by granulation.

Gross appearances: If the infection is confined at first to the lymph vessels (lymphangitis) dark red lines appear, extending from the infected wound along the line of lymph vessels. The lymph nodes along the line of the lymphatics become infected, enlarged, and are tender. If the infection persist, in the course of a few hours the tissue becomes diffusely reddened and swollen, and is tender and pits on pressure. The lymph nodes may break down and form an abscess at a considerable distance from the original point of infection.

If the infection (primarily) extends along the connective tissue septa, redness and swelling are seen about the infected wound, and the skin pits on pressure. This area of exudation enlarges peripherally, and may extend over enormous areas. If the infection is very severe, blebs and bullæ filled with pus appear on the surface. In certain cases the superficial layers of the skin are involved, and skin and subcutaneous tissue may be cast off as a slough, leaving a dirty grayish surface behind. In cases which do not go on to ulceration, if the tissue is incised during the acute stage a thin fluid escapes, while the subcutaneous tissue is yellow, gelatinous, and the skin is porky.

The process may quiet down in a few days, leaving the skin reddened, firm, and adherent to the underlying tissues from the

formation of granulation tissue. In severer forms, after the slough is separated, a more or less extensive granulating wound is left, which heals like other granulating wounds.

Erysipelas is an acute inflammation of the skin and subcutaneous tissue, usually due to infection of large or microscopic wounds. Rarely it may be due to infection of the hair follicles. The process is due to infection by a streptococcus, probably identical with the streptococcus pyogenes which produces phlegmon. The process in general is the same as that in phlegmon, but the infection generally is more superficial.

Gross appearances: Erysipelas appears as a localized redness, glazing, and swelling of the skin. The exudation may persist for a time in one place and then extend to adjacent regions. In severe cases blebs and pustules may appear on the surface, and portions of the skin may become necrotic and gangrenous. As a rule, the skin returns to its normal condition, and the formation of connective tissue is slight, unless necrosis and gangrene have occurred.

Principles of treatment.

Abscess: In some cases of acute superficial abscess of the skin it is possible to "abort" the abscess by injection of strong aseptic fluid, *e.g.*, carbolic acid. In all abscesses the chief essential is opening and drainage of the purulent fluid. In carbuncles complete excision often is the best method of treatment.

Phlegmon: Incision and drainage are indicated.

Erysipelas: On account of the wandering character of the disease incision is useless.

OSTEOMYELITIS

is acute suppuration of bone, due to infection by pathogenic microörganisms, generally the staphylococcus aureus. It has been designated "furunculosis" of bone.

Cause: The staphylococcus aureus is the organism most commonly present. Sometimes other organisms, streptococcus, pneumococcus, and typhoid bacillus, produce the disease. Certain factors predispose to the disease. It occurs generally in young adults or children. Exposure to cold and fatigue often precede the attack. The disease frequently occurs subsequent to acute infectious disease, *e.g.*, typhoid fever, scarlet fever, etc. In these cases the process generally is due to a secondary infection

with pyogenic organisms, but sometimes is due to primary infection by the specific organism, *e.g.*, "typhoid osteomyelitis." The disease often is secondary to suppuration elsewhere, *e.g.*, furuncle. Frequently after compound fractures the injured ends are infected by microörganisms, and "traumatic osteomyelitis" results.

Process: The infection always begins in the marrow of bone. The trabeculæ and cortical portions are destroyed secondarily. The disease begins almost always in the diaphysis of a bone, often near the epiphyseal line. The process may begin rarely in the epiphysis and extend to and destroy the joint.

If the organisms obtain access to the marrow they produce a toxin which causes necrosis of marrow cells. Since the marrow spaces communicate freely with each other the toxic material is retained, and so necrosis may be very extensive before infiltration with exudate is marked. The marrow tissue then appears necrotic over large areas, and throughout the necrosis are numerous colonies of cocci. Exudation follows, and is chiefly leucocytes. Solution of marrow cells, often over considerable areas, may occur. Sometimes there is more or less solution of bony trabeculæ, so that a cavity is formed containing leucocytes and tissue detritus, "bone abscess." The process may extend along the marrow spaces and involve the marrow of the entire shaft in a very short time. The infection generally extends through cortical bone to the periosteum. In that case the periosteum may be stripped and elevated from the bone over a large area. Between the bone and periosteum a purulent exudate is present, often in considerable amount. The infection may extend to the soft tissues and cause necrosis and suppurative inflammation, and result in the formation of an abscess, often very extensive, connecting with the bone. The abscess may extend to the surface, involve the skin, and open and discharge through one or more sinuses. The process usually does not extend beyond the epiphyseal line, but may cause separation of the epiphysis (spontaneous fracture), or even extend to and destroy the epiphysis. I the epiphysis is involved the infection may extend to the joint cavity, produce suppuration and destruction of the joint. General infection, toxæmia, and metastasis may occur early in the disease.

Repair: The reparative process which follows suppuration produces certain peculiar gross conditions. In the marrow spaces granulation tissue forms, is converted into scar tissue, and walls

off the necrotic portion from the healthy portion of the shaft. The periosteum becomes separated from the underlying necrotic shaft (sequestrum) and forms a layer of new periosteal bone, which surrounds the original shaft as a more or less complete shell (involucrum).

Proliferation of the connective tissue of the marrow begins in a few days. Since the marrow spaces open out into each other in an irregular way a microscopic section may show adjacent spaces, in one of which is necrotic tissue and exudate, while an adjoining one is filled with granulation tissue. Granulation tissue may fill up marrow spaces and attempt to encapsulate the inflamed area, or, if one entire end of the shaft is necrotic, may extend across the shaft and form a layer of granulation tissue separating the necrotic from the sound portion of the shaft. The granulation tissue shows extremely numerous lymphoid and plasma cells. In time the granulation tissue becomes dense scar tissue, separating the necrotic, loosened end of the shaft. If the entire diaphysis has been involved in the original, granulation tissue may develop in some portions and not in others, so that some of the marrow spaces are filled with exudate, others with granulation tissue, giving a peculiar mottled appearance of yellow and red to the bone on section. Walling off of the necrotic shaft may take from two to several months.

The periosteum proliferates early. At first it forms a thickened layer like granulation tissue. In the course of a few weeks new trabeculæ appear, like those in the external callus of a fracture (see Fractures). By the end of eight to sixteen weeks this layer of new periosteal bone is of considerable thickness ($\frac{1}{16}$ inch). In the course of months this bone thickens, becomes bone like cortical bone, and surrounds the original shaft like a shell "involucrum," and becomes separated from the original shaft, which also is separated from the healthy portion of the shaft. The loose fragment of the original shaft may persist as a "sequestrum," and lies in a cavity lined with granulation tissue, forming an open sore which may discharge for years; or the sequestrum may be disintegrated and discharged piecemeal through sinuses. If the sequestrum is removed, artificially or naturally, it leaves a cavity lined with granulation tissue, surrounded by dense cortical bone, which shows very little tendency to heal, and may persist for years.

Gross appearances: At first the infected bone may show no

visible change beyond intense local pain. If the process extends to the periosteum and soft tissues it produces enormous deep swelling with redness and pitting on pressure. If the abscess extend towards the surface, fluctuation may appear. The abscess may point at one or more places and break through the skin and discharge its contents through one or several sinuses. After evacuation of the contents of the abscess the shaft appears nearly of normal thickness. In a few weeks the shaft appears appreciably greater in thickness, due to the formation of the periosteal shell. At the end of twelve to sixteen weeks the periosteum is about $\frac{1}{16}$ of an inch thick and contains thin plates of periosteal bone. In a few weeks more (sixteen to thirty weeks) the periosteal shell becomes thick enough to take on the function of the original shaft. The original shaft during this time may be felt as a shaft of bare bone. In time it becomes loosened and may be removed by operation, or may be discharged spontaneously through sinuses.

At any of these stages, *i.e.*, early necrosis, early periosteal proliferation, or in the stage of involucrum and sequestrum, it is possible to take advantage of the regenerative power of the periosteum and endosteum to bring about complete regeneration of bone.

In all cases it first is necessary to remove the necrotic bone, which acts as a foreign body. After the necrotic bone is removed the intact periosteum should be approximated so as to bring the internal surfaces together and to leave no central cavity. The growth of the periosteum is peripheral, and new bone, like the external callus, is formed, until a shaft of periosteal bone is produced which slightly exceeds in size that of the original shaft. As the bone becomes harder as it grows older, there is some absorption of the bone, until ultimately the new bone is of the same size as the original shaft. The new bone at first is solid bone without a marrow canal, but finally, judging from X-ray pictures, there is an absorption of the bone in the centre of the shaft, and a new marrow canal is formed. The notable thing about this process of bone regeneration by the periosteum is that the new bone is of exactly the same shape as the original bone, and cannot be distinguished even by touch, sight, or the X-ray. This suggests that the shape of the bones of the human skeleton is due to two causes — heredity, and environment or function. Hence, when a bone is removed the new bone which is formed is of the

shape which performs function to the best advantage. This is true of very complicated bones, and even of complicated joints which are excised subperiosteally.

In some cases where the involucrum is old it has limited power of repair and, in such cases, both involucrum and sequestrum must be removed to give the periosteum a chance to form an entirely new bone.

This theoretically ideal method of repairing destruction caused by acute suppuration in bone is not always practicable for various surgical reasons. In some cases it is better to employ aseptic blood clot, or osteoplastic bone flaps, or curettage and skin grafting.

APPENDICITIS.

Acute suppuration of the vermiform appendix is the most common cause of peritonitis in men, and is a frequent cause of peritonitis in women.

Etiology: The process probably is due to infection by pyogenic organisms of a superficial necrosis of the mucous membrane of the appendix. The cause of the primary necrosis frequently is fæcal concretions, rarely foreign bodies (*e.g.*, pin, intestinal parasites, fish bones, etc.). The organism that infects the necrosis is in 90 per cent. of the cases the streptococcus pyogenes. Cultures taken from appendiceal abscesses always show a profuse growth of the colon bacillus, and pyogenic organisms may not develop. If fresh smears from the abscess are examined, however, streptococci, frequently dead, are found. Pure cultures of the colon bacillus injected into the peritoneum of animals do not cause peritonitis. So it is probable that the original infection in these cases is due to the streptococcus, and the organism does not appear in cultures because it is crowded out by the resistant colon bacillus.

Process: Superficial necrosis or lowered resistance of appendiceal mucous membrane is caused by fæcal concretions or other foreign bodies. Infection by pyogenic organisms, generally the streptococcus, takes place. The infection produces necrosis and acute exudation in the wall of the appendix. The infection may extend through the wall of the appendix, and cause a localized infection of the adjacent peritoneum, without perforation; usually the necrosis produces sloughing and perforation of the wall of the appendix. The perforation may be small or large, and occur at

the tip or near the cæcal attachment; or the entire appendix may become gangrenous and slough. Perforation of the appendix allows extravasation of intestinal contents and a local or general infection of the peritoneum (extravasation peritonitis).

If the perforation is small and takes place near the tip the amount of extravasation usually is small, and the resulting peritonitis is circumscribed. As a result of the peritonitis a fibrinous or fibrino-purulent exudate is formed upon the surface of the appendix and intestines. This exudate binds together folds of adjacent intestine, and may "wall off" the perforated appendix from the general peritoneal cavity (appendiceal "cake"). An abscess may form about the appendix, surrounded by coils of adherent intestine. Such an abscess may burst through the adhesions and infect the general peritoneal cavity. If the process subsides fibrinous adhesions become organized and fibrous bands fasten coils of intestine firmly together. The exudate upon the surface of the appendix may become organized and increase the size of the organ to an enormous extent.

If the perforation is large and near the cæcal attachment, or if the entire appendix becomes gangrenous, a very extensive extravasation of fæcal contents may take place in a very few hours. This may take place so rapidly that adhesions may be unable to wall off the appendix and a general infection of the peritoneal cavity may occur. Such cases of general peritonitis usually are fatal.

During the acute stage the mucous membrane of the appendix shows more or less extensive areas of necrosis, exudation, and ulceration. The lumen of the appendix is filled with purulent exudate. All the coats of the appendix are infiltrated with exudate. Upon the serous surface is an exudate, largely fibrinous. If the attack subside the ulcer heals, the walls become thickened by formation of granulation tissue, and infiltrated with lymphoid and plasma cells. The exudate on the serous surface is replaced by granulation tissue. In time the walls are thickened from the presence of dense fibrous tissue and the appendix is surrounded by a more or less extensive layer of dense fibrous tissue.

Gross appearances: During the early hours the appendix is injected and swollen from the presence of exudate. On the surface is a layer of exudate. The wall may show a small or large perforation. About the appendix may be adherent coils of injected intestine; or an abscess, often very extensive, filled with

foul yellow or hæmorrhagic pus, forms about the appendix, shut off from the general peritoneal cavity by folds of inflamed intestine. If the process is very severe the appendix may slough *en masse* and lie in the abscess cavity, or entirely disappear. If the attack subside the appendix, much thickened by organized tissue, may be surrounded by folds of intestine, firmly bound together by bands of granulation tissue, which in time become dense fibrous tissue. If infection of the general peritoneal cavity take place during the acute attack, all abdominal viscera are injected, covered with a very extensive layer of fibrino-purulent or purulent exudate, yellow, green, or hæmorrhagic. Much free pus may collect in certain portions of the abdomen. Gas often is present.

Tubercular appendix: In tuberculosis of the intestinal tract the appendix may become infected. Tubercular ulceration may cause perforation of the appendix with extravasation of fæcal contents, but is very rare.

Characteristic tubercular lesions are seen in the wall of the appendix.

Typhoid appendix: Occasionally in typhoid fever ulceration and perforation of the appendix occurs. Very rare.

Principles of treatment: In some cases it is possible to remove a diseased appendix before acute suppuration occurs. If acute suppuration does occur it is to be treated like other abscesses, *i.e.*, by drainage, with removal of the appendix. In some cases if the acute suppuration subsides it is possible to remove the appendix during the quiescent stage, *i.e.*, "between the attacks." If general infection of the peritoneal cavity occurs it must be treated like any general peritonitis.

TUBERCULOSIS OF THE BONES AND JOINTS.

Cause: Infection of the marrow of the bone by the tubercle bacillus. The identity of the disease with other tuberculous tissues is proven by the presence of miliary tubercles containing the tubercle bacillus, by inoculation of animals with bone, soft tissue from joints or wall of a cold abscess, producing tuberculosis in susceptible animals, and by the experimental production of joint lesions in animals, similar to those produced in human beings. Certain causes favor the occurrence of tuberculosis of bone. It occurs chiefly in early youth. Trauma of moderate severity, *e.g.*, sprains, often precedes joint lesions. Tuberculous joints usually are secondary to old tuberculous foci and tubercular

disease elsewhere in the body; *e.g.*, bronchial or mesenteric lymph nodes. Primary disease of the bones occurs rarely; of the joints, almost never.

The tubercle bacillus, having obtained access to the bone marrow, causes the formation of miliary tubercles which arise by proliferation of the connective tissue of the marrow. As the tubercles enlarge, the centre becomes caseous, and by fusion of caseous areas an area of softening is produced in the bone. The trabeculæ at first are not involved, but at length may soften and break down, forming a cavity (tuberculous abscess) in the bone.

Bones: In the *long* bones tuberculous disease begins almost invariably in the marrow of an epiphysis, never in the cartilage, and very rarely in the diaphysis. By coalescence and caseation of tubercles the marrow is destroyed, often in considerable areas. If the trabeculæ in such an area dissolve, a cavity is formed (tuberculous bone abscess). In other cases the trabeculæ retain their shape, while the marrow spaces are filled with tuberculous necrotic tissue; *i.e.*, a "tuberculous sequestrum" is formed. In the marrow spaces surrounding either abscess or sequestrum is a formation of granulation tissue. The tuberculous area enlarges peripherally, extends towards the surface, and may open upon the surface (very rare) or into the adjacent joint (usual).

In the *short long* bones, *e.g.*, the phalanges, the process may begin in the diaphysis. The entire shaft may become tuberculous, while the periosteum proliferates, as in the case of osteomyelitis (*q.v.*), and forms a new bony shell. As a result, the shaft enlarges, becomes spindle-shaped, while the tuberculous process may open upon the surface by various sinuses (spina ventosa).

In the *short* bones the tubercles often destroy an entire bone, and secondarily invade several adjacent bones and joints at a comparatively early period.

Tuberculous disease of the flat bones is less frequent, but not uncommon. The cranial bones frequently are attacked. No bone in the body is exempt.

General miliary tuberculosis of the bones occurs in cases of acute miliary tuberculosis. In that case the miliary tubercles are seen scattered throughout the marrow, but they never attain a size sufficient to cause clinical symptoms.

Abscess: "Cold Abscess." If tubercular disease extends from the bones to the adjacent soft tissues, tubercles form, enlarge, become caseous, and coalesce and form a cavity which contains

necrotic tuberculous material, serum, and comparatively few leucocytes ("cold abscess"). About such an abscess is a layer of tuberculous tissue, composed either of discrete miliary tubercles or of a layer of diffuse ulcerating tuberculous tissue. Surrounding this tuberculous tissue is a layer of reactionary granulation tissue. Such an abscess may open upon the surface of the body by a sinus which connects with the abscess cavity. The lining of the sinus is tuberculous tissue, like that which lines the abscess.

Joints: Primary tuberculous disease of the joint occurs seldom or never, and tuberculosis of the joint almost invariably is secondary to tuberculous disease of the adjacent epiphysis. As the tubercular focus in the epiphysis enlarges, it extends towards the cartilage which covers the end of the bone. The cartilage becomes fibrillated and finally perforated at one or more points. The tubercle bacillus is diffused throughout the synovial fluid. The tubercle bacilli are taken into the lymphatic spaces of the synovial membrane and produce miliary tubercles. By enlargement, fusion, and caseation of these tubercles the surface of the synovial membrane becomes covered with tuberculous ulcers. From the attachment of the synovial membrane to the cartilage the tuberculous tissue extends over and beneath the cartilage which covers the ends of the bones (tuberculous "pannus"). Wherever the cartilage comes in contact with this tuberculous tissue it becomes fibrillated and ultimately destroyed. As a result, the cartilage may be perforated at various points, or lifted from the underlying bone and finally completely destroyed. The bone beneath the destroyed cartilage may become involved secondarily. The tuberculous process may extend along the ligaments and destroy them. Ultimately there is destruction of the synovial membrane, cartilage, and articular ends of the bone, which results in luxation or partial dislocation. The synovial cavity of a tuberculous joint contains exudate and necrotic tubercular tissue. It may vary from serous to sero-purulent.

Repair: If the tuberculous process cease, granulation tissue replaces the destroyed tissue. This granulation tissue may be transformed into fibrous tissue, and produce adhesions between adjacent joints (fibrous anchylosis). Or the fibrous tissue may be converted (metaplasia) into cartilage (cartilaginous anchylosis). True bone may form in the cartilage (bony anchylosis). Generally the process is the same in all the bones and joints. Certain peculiarities occur in bones and joints of special regions.

Spine: Tuberculous disease begins in the marrow of the anterior portion of the body of the vertebræ, and causes tuberculous softening of the affected parts. The process may extend forward and beneath the prevertebral ligaments. In that way the body of adjacent vertebræ are involved. As the vertebræ soften, the superincumbent weight causes a bending forward of that portion of the spine above the disease, and an angular deformity or "knuckle" appears in the back. The tubercular process may extend to the soft tissues in front of the spine and produce a tubercular abscess ("cold abscess"). If the disease is in the cervical region this abscess may project into the pharynx ("retropharyngeal abscess"). If the abscess is lower it may extend into the pleural cavity, and has been known to rupture into the lungs or large vessels. Or the abscess may first appear in the back, outside of the quadratus muscle ("lumbar abscess"). Or it may appear in the ilio-psoas region ("iliac abscess"). Such an abscess occasionally ruptures into the intestines. Or it may extend beneath Poupart's ligament ("femoral abscess").

Secondary distortion of the bones due to malposition and pressure occurs. It is said that alteration in the diameters of the skull occurs. Deformities of the ribs and thorax are common ("pigeon breast," etc.).

Various deformities of the large vessels may occur, especially if the disease arises rather late. Deformities are due to the fact the spine is shortened, while the blood vessels retain their length.

Pott's paralysis: The tuberculous process may extend backwards through the bodies of the vertebræ to the spinal dura. Tuberculous granulations may surround and cause pressure upon the spinal cord. Pressure upon the cord never is caused by diminution of the calibre of the spinal canal, due to the formation of the knuckle. Occasionally the pressure is due to loose fragments of tuberculous bone. The pressure causes paralysis of the cord below the point of pressure ("Pott's paralysis"). Destruction of the cord rarely follows, and most cases of Pott's paralysis regain complete function.

If the tubercular process cease the lost tissue is replaced by fibrous tissue, which ultimately may be converted into bone and lead to a more or less complete anchylosis of several adjacent vertebræ. In these cases the knuckle becomes rounded, rather than angular. Large abscesses may be inspissated or absorbed, or their contents may become calcified.

Hip: The disease begins sometimes in the upper epiphysis of the femur, very frequently in the acetabulum. If it begin in the epiphysis it usually extends into the joint, erodes and destroys the head of the femur. The acetabulum is softened, and under pressure may be extended upwards ("wandering acetabulum"). Extension to the soft tissues and formation of an abscess is common. If the acetabulum is the primary seat of the disease destruction of the joints follows. The process, however, frequently extends through the base of the acetabulum, and an abscess is formed within the pelvis. Acetabular disease, as a rule, therefore, is much more serious than hip disease, which begins in the epiphysis.

If the head of the femur is extensively destroyed partial or complete dislocation of the head of the bone may occur. Occasionally the process causes fracture of the neck of the femur. The contraction of certain muscles leads to characteristic deformity of the leg. Often early shortening of the leg occurs either from partial dislocation or from failure of the bone to develop at the usual rate.

Knee: "Tumor Albus." The process in the knee may begin in any of the bones which enter into the knee joint. Primary disease of the petalla is not uncommon. Rarely the fibula is primarily affected. The destruction of the joint often is rapid, and secondary muscular contraction draws the lower leg upward (flexion), outward, and backward. Enormous swelling of the knee usually occurs. Abscesses and sinuses are common.

Ankle joint: The disease may begin in any of the bones of the tarsus. If one small bone is affected primarily several adjacent small bones usually are involved, so that extensive destruction is required to remove the entire process. Abscess and sinus formation is common.

Other joints: The wrist, elbow, and shoulder joints are less frequently diseased than the joints of the leg, but are not uncommon. The cranial bones, sternum, clavicle, and ribs are affected still less often.

Gross appearances: Bone. Recognition of the limits of tuberculous disease at the time of operation is difficult. The bone may show either more or less extensive cavities filled with caseous material, surrounded by purplish granulation tissue, or may show softened yellowish masses of bone, surrounded by granulation tissue. Miliary tubercles seldom can be recognized.

Joints: If the synovial membrane is affected it shows at first a surface studded with discrete yellowish or whitish circular areas, somewhat variable in size, not larger than the head of a pin. If the process has persisted the surface of the synovial membrane is pitted with more or less extensive ulceration. Occasionally in the joints are extensive papillary growths, "fungus joints," composed of tuberculous tissue. The amount of fluid in the joints is variable — it may be very slight ("caries sicca"), or it may be large, and consist of cloudy serum ("hydrops"), or of fluid, caseous material and pus, into which recent or remote hæmorrhages may have taken place.

Principles of treatment: It is to be remembered that in many cases tuberculosis of bones and joints is a self-limited disease. To favor this spontaneous cure of the disease it is necessary to keep the diseased tissues at rest and protected. This is accomplished in various ways in different parts of the body. Various braces and splints are employed, *e.g.*, Taylor back brace, Taylor hip splint, Thomas knee splint, etc. In some cases plaster of Paris splints are useful, plaster jackets, etc. In other cases rest in bed with fixation is employed (Bradford frame, etc.). The ideal method would be to detect the tubercular focus while it is confined to the bone, by the X-ray, and to excise the entire affected area. This seldom is practicable. In many cases palliative operations are indicated on general surgical grounds.

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